In the Specification:

Please replace the paragraphs begin at page 1, line 19, end at page 2, line 18, with the following substitute paragraph:

The observation that vasoactive intestinal peptide (VIP) is cleaved by antibodies (Abs) from asthma patients provided early evidence that Abs may possess peptidase activity [1,2]. This observation has been reproduced independently by Suzuki et al [3]. Autoantibody catalysis is not restricted to catalysis of VIP. Autoantibodies in Hashimoto's thyroiditis catalyze the cleavage of thyroglobulin [4]. Further evidence for autoantibody catalysis has been provided by reports of DNase activity in Abs from lupus patients [5,6]. The bias towards catalytic Ab synthesis in autoimmune disease is supported by observations that mouse strains with a genetic predisposition to autoimmune disease produce esterase Abs at higher levels when compared to control mouse strains in response to immunization with a transition state analog [7].

Like noncatalytic Abs, peptidase Abs are capable of binding Ags with high specificity mediated by contacts at residues from the VL and VH domains. The purified H and L subunits are known to be independently capable of binding antigens (Ags), albeit with lower affinity than the parent Ab. X-ray crystallography of Ab-Ag complexes have shown that the VL and VH domains are both involved in binding the Ag [8]. The precise contribution of the two V domains varies in individual Ab-Ag complexes, but the VH domain may contribute at a somewhat greater level,

because CDRH3 tends to be longer and more variable in sequence compared to CDRL3.

Please replace the section title at page 105, lines 5-7, with the following substitute title:

Use of CRAAs and Catalytic Antibodies in Ischemia-reperfusion Injury and Septic Shock/SIRS
~~**Larry I Need the References for This Section**~~

Please replace the paragraph at page 8, lines 24-32 with the following substitute paragraph:

FIG. 1 is a free energy diagram for antibody catalysis involving stabilization of the substrate ground state (ΔG_S) and transition state (ΔG_{TS}). $\Delta G^{\dagger}_{uncat}$ and $\Delta G^{\dagger}_{cat}$ correspond to activation energies for the uncatalyzed and catalyzed reactions, respectively. Km is a function of the extent of ground state stabilization (ΔG_S). Kcat/Km is a function of the extent of transition state stabilizatin relative to the catalyst-substrate ground state complex. $\underline{\Delta G_p \text{ is the product ground state.}}$